

I claim:

1. A method for treating movement disorders comprising:

administering an effective dose of an agent that increases GABA-A

5 neurotransmission and decreases NMDA-glutamate neurotransmission to a patient with a movement disorder.

2. The method of claim 1, wherein said movement disorder is selected from the group consisting of simple tics, multiple tics, Tourette's syndrome, focal dystonias, blepharospasm, and
10 Meige syndrome.

3. The method of claim 1, wherein said agent is selected from the group consisting of acamprosate (calcium N-acetylhomotaurinate), magnesium N-acetylhomotaurinate, lithium N-acetylhomotaurinate, salts of N-acetylhomotaurinate, acetylhomotaurinate base and derivatives thereof that share the pharmacodynamic effects of acamprosate on GABA and glutamate transmission by enhancing GABA-A transmission and reducing NMDA-type glutamate transmission.

4. The method of claim 1, wherein said agent is available in the blood.

5. The method of claim 1, wherein said agent is available in the brain.

6. The method of claim 1, wherein said agent is a pro-drug metabolized in the body to release acetylhomotaurinate ion into the body

7. The method of claim 6, wherein the agent released into the body is selected from the group consisting of any derivative of homotaurinate or acetylhomotaurinate with similar pharmacodynamic effects on GABA and glutamate neurotransmission as acetylhomotaurinate.

8. The method of claim 6, wherein said pro-drug is metabolized in the liver, blood or brain.

9. The method of claim 6 wherein, said pro-drug comprises an ester of acetylhomotaurinate or a derivative of homotaurine or acetylhomotaurine with similar pharmacodynamic effects on GABA or glutamate transmission.

10. The method of claim 1, wherein said agent comprises a derivative of calcium acetylhomotaurinate, homotaurine or acetylhomotaurine with similar pharmacodynamic effects on GABA or glutamate transmission.

11. The method of claim 10 wherein said derivatives have a longer half life than acamprosate.

12. The method of claim 10, wherein said derivatives are absorbed better from the gastrointestinal tract.

13. The method of claim 10, wherein said derivatives are absorbed more reliably from the gastrointestinal tract.

14. The method of claim 1, wherein treating the movement disorder reduces cognitive symptoms of the movement disorder when said symptoms are determined by subjective performance, mental status examination, or through the use of neuropsychological tests.

15. The method of claim 1, wherein said step of administering comprises oral administration.

16. The method of claim 1, wherein said movement disorder is related to a deficiency in GABA in the basal ganglia.

17. The method of claim 1, wherein said movement disorder is related to glutamate-based excitotoxicity.

18. A method for treating movement disorders comprising the steps of:

5 selecting a first pharmacologically active agent that acts as a GABA-A receptor agonist and selecting a second pharmacologically active agent that acts as a NMDA-type glutamate receptor antagonist; and

administering said first and said second agents to a patient with a movement disorder.

10 19. The method of claim 18, wherein the movement disorder is selected from the group consisting of simple tics, multiple tics, Tourette's syndrome, focal dystonias, blepharospasm, and Meige syndrome.

20. The method of claim 19, wherein the blepharospasm is idiopathic blepharospasm.

21. The method of claim 19, wherein the blepharospasm is associated with a neuroleptic-induced movement disorder.

22. The method of claim 18, wherein the step of administering comprises selecting dosages of the first and second agents such that the administration of said first and second dosages reduces symptoms of said movement disorder at non-toxic.

23. The methods of claims 18, wherein the step of selecting, said first agent and said second agent are the same agent.

24. The method of claim 18, wherein said movement disorder comprises involuntary movements similar to those seen in Tourette's syndrome, focal dystonias, blepharospasm and

tics.

25. The method of claim 18, whereby said movement disorder is associated with Huntington's disease.

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26. The method of claim 18, wherein treating Tourette's syndrome, focal dystonias, blepharospasm or tics reduces cognitive symptoms associated with the movement disorder when said symptoms are determined by subjective report, by mental status examination, or through the use of standard neuropsychological tests.

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27. The method of claim 18, wherein said movement disorder is related to a deficiency in GABA in the basal ganglia.

28. The method of claim 18, wherein said movement disorder is related to glutamate-based excitotoxicity.

29. The method of claim 18, wherein the step of selecting further comprises selecting a third pharmacologically active agent that is a noncompetitive NMDA receptor antagonist, or an ion channel blocker at channels linked to NMDA receptors.

30. The method of claim 18, wherein the step of administering further comprises administering said third agent in conjunction with said first and said second agents.

31. The method of claim 30, wherein said third agent is memantine.

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32. The method of claim 31, wherein said third agent is a derivative of memantine with similar pharmacodynamic effects at NMDA receptors.

33. The method of claim 30, wherein said third agent is magnesium.

34. A method for assessing risk of developing a neuroleptic or dopamine receptor blocker-induced movement disorder comprising:

5 performing standard tests of total body magnesium status.

35. The method of claim 34 comprising, measuring magnesium retention after an oral or parenteral magnesium load.

10 36. The method of claim 34, wherein the step of administering comprises selecting a dose of magnesium that is sufficient for evaluating magnesium retention and is non-toxic.

37. A method for preventing neuroleptic or dopamine receptor blocker-induced movement disorders comprising:

reducing the risk the movement disorders by administering to a patient at risk for developing a movement disorder, an effective dose of magnesium ion.

38. The method of claim 37, further comprising:

delaying the onset of the neuroleptic or dopamine receptor blocker-induced movement disorder by administering to a patient at risk for developing a movement disorder, an effective dose of magnesium ion.

39. A method of treating movement disorders comprising:

25 reducing the symptoms of the movement disorder by administering to a patient at risk for developing a movement disorder, an effective dose of magnesium ion.

40. The method of claim 39, further comprising:

augmenting the therapeutic effects of NMDA receptor antagonists and down-regulators in patients with movement disorders by administering to said patient an effective dose of magnesium ion.

41. The method of claims 37 or 39, wherein said movement disorder is selected from the group consisting of tics, multiple tics, Tourette's syndrome, focal dystonias, blepharospasm, and Meige syndrome.

42. The method of claim 39 further comprising:

administering to a patient with a movement disorder an effective dose of magnesium N-acetylhomotaurine sufficient to decrease the symptoms of the movement disorder.

43. The method of claim 42, wherein the magnesium N-acetylhomotaurine administered is a magnesium salt of any derivative of N-acetylhomotaurine that shares its property of enhancing GABA-A neurotransmission and attenuating NMDA-glutamate neurotransmission.

44. The method of claim 42, wherein the step of administering comprises administering the magnesium salt of any derivative of N-acetylhomotaurine that is an effective treatment.

45. A method for treating a movement disorder comprising:

administering to a patient in combination, a single pill at an effective dose,

- (i) an NMDA receptor antagonist
- (ii) a GABA-A agonist
- (iii) magnesium ion.

46. The method of claim 45, wherein the NMDA receptor antagonist and the GABA-A agonist is the same agent.

47. The method of claim 45, wherein the magnesium ion is in the form of a magnesium salt.

48. The method of claim 45, wherein the NMDA antagonist and the GABA-A agonist is selected from the group consisting of acamprosate (calcium N-acetylhomotaurinate), magnesium
5 N-acetylhomotaurinate, salts of N-acetylhomotaurinate, acetylhomotaurinate base, homotaurine and derivatives thereof with similar pharmacodynamic effects upon GABA and glutamate neurotransmission.

49. The method of claim 48, wherein said derivative is available in the blood.

50. The method of claim 48, wherein said derivative is available in the brain.

51. The method of claim 48, wherein said derivative is a pro-drug metabolized in the liver, blood, or brain, to release acetylhomotaurinate ion.

52. The method of claim 48, wherein said derivative is a pro-drug metabolized in the liver, blood, or brain to release any derivative ion of acetylhomotaurinate ion with similar pharmacodynamic effects on GABA and glutamate neurotransmission.

53. The method of claim 48, wherein, said pro-drug comprises an ester of acetylhomotaurinate, or any derivative of acetylhomotaurine or homotaurine with similar pharmacodynamic effects on GABA and glutamate neurotransmission

54. The method of claim 45, wherein said derivative has a longer half -life than acamprosate.

55. The method of claim 45, wherein said derivative is absorbed from the gastrointestinal tract better than acamprosate.

56. The method of claim 45, wherein said derivatives are absorbed better from the gastrointestinal tract.

57. The method of claim 45, wherein the pill is used to treat Tourette's syndrome.

58. The method of claim 45, wherein the pill is used to treat multiple tics.

59. The method of claim 45, wherein the pill is used to treat simple tics.

60. The method of claim 45, wherein the pill is used to treat blepharospasm or Meige syndrome.

61. The method of claim 45, wherein the pill is used to treat focal dystonias.

62. The method of claim 45, wherein an effective dose of

- (i) an NMDA receptor antagonist
- (ii) a GABA-A agonist
- (iii) magnesium ion

is delivered in the form of delivery agent comprising a syrup, an elixir, a liquid, a tablet, a time-release capsule, an aerosol or a transdermal patch.

63. A pill for treating movement disorders comprising:

one or more agents that increase GABA-A neurotransmission;
one or more agents that decrease NMDA-glutamate neurotransmission; and
magnesium ion.

64. The pill of claim 63, wherein the NMDA receptor antagonist and the GABA-A agonist is the same agent.

65. The pill of claim 63, wherein the magnesium ion is in the form of a magnesium salt.

66. The pill of claim 63, wherein the NMDA antagonist and the GABA-A agonist is selected from the group consisting of acamprosate (calcium N-acetylhomotaurinate), magnesium N-acetylhomotaurinate, salts of N-acetylhomotaurinate, acetylhomotaurinate base, homotaurine and derivatives thereof with similar pharmacodynamic effects upon GABA and glutamate neurotransmission.

67. The pill of claim 66, wherein said derivative is available in the blood.

68. The pill of claim 66, wherein said derivative is available in the brain.

69. The pill of claim 66, wherein said derivative is a pro-drug metabolized in the liver, blood, or brain, to release acetylhomotaurinate ion.

70. The pill of claim 66, wherein said derivative is a pro-drug metabolized in the liver, blood, or brain to release any derivative ion of acetylhomotaurinate ion with similar pharmacodynamic effects on GABA and glutamate neurotransmission.

71. The pill of claim 66, wherein said pro-drug comprises an ester of acetylhomotaurinate or a related compound with similar pharmacodynamic effects on GABA and glutamate neurotransmission.

72. The pill of claim 63, wherein said derivative has a longer half life than acamprosate.

73. The pill of claim 63, wherein said derivative is absorbed from the gastrointestinal tract better than acamprosate.

74. The pill of claim 63, wherein said derivative is more reliably absorbed from the gastrointestinal tract.

75. The pill of claim 63, wherein the pill is used to treat Tourette's syndrome.

76. The pill of claim 63, wherein the pill is used to treat simple tics.

77. The pill of claim 63, wherein the pill is used to treat multiple tics.

78. The pill of claim 63, wherein the pill is used to treat blepharospasm.

79. The pill of claim 63, wherein the pill is used to treat focal dystonias.

80. The pill of claim 63, wherein an effective dose of

(i) an NMDA receptor antagonist

(ii) a GABA-A agonist

(iii) magnesium ion

is delivered in the form of delivery agent comprising a syrup, an elixir, a liquid, a tablet, a time-release capsule an aerosol or a transdermal patch.

81. A composition comprising at least two agents, the composition having activities of:

(i) enhancing GABA-A neurotransmission

(ii) decreasing NMDA-type glutamate neurotransmission.

82. The composition of claim 81, wherein said composition is a compound.

83. The composition of claim 81, wherein said composition is a mixture.

84. The composition of claim 81 wherein neither agent has both activities.

85. A composition comprising:

- (i) acamprosate
- (ii) an inorganic salt or chelate of magnesium.

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86. The composition of claim 85, wherein the ratio of acamprosate to the inorganic magnesium salt or chelate is between 1:1 and 6:1 by weight.

87. The composition of claim 85, wherein the inorganic salt or chelate of magnesium is any
10 inorganic salt or chelate of magnesium.

88. The composition of claim 85, wherein the inorganic salt or chelate of magnesium comprises magnesium chloride, magnesium oxide, magnesium sulfate, and magnesium chelated with any of various amino acids.

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